sharply. Indeed there was one fewer in 1963 than in 1953, though there had been some increase in 1961 and 1962. The 10 years' operative mortality was 0.87% and the morbidity due to chest infection, wound infection, and deep venous thrombosis was 9.8%.

I can confirm the finding of a high proportion of stones at operation (97.4% in 1960), whereas only 29% had been demonstrated radiologically preoperatively in that year. The most striking feature of 1960 was that there were no operative cholangiograms carried out. Indeed, 1961 was the first year they were done at Frenchay, though only 11 were done in the subsequent three years. This led to 37.6% of all common bile ducts being explored, half of these unnecessarily. As operative cholangiography is now routine during all cholecystectomies at that hospital it would be interesting to compare the 1960 and 1970 total figures with special reference to the problem of recurrent common-bile-duct stones.—I am, etc.,

R. G. WILSON

Royal Infirmary,
Edinburgh

Gas Gangrene and Hyperbaric Oxygen

SIR,—Your leading article (23 September, p. 715) implies that the case has been proved for the treatment of gas gangrene with hyperbaric oxygen on the basis of sound experimental and clinical evidence. Equally, however, it could be argued that the evidence is somewhat equivocal.

Various experimental studies¹⁻⁴ have shown that hyperbaric oxygen is of no value in the treatment of established gas gangrene, and in man antibiotics in conjunction with either conservative or aggressive surgery have yielded survival rates^{5,6} which are better than those quoted for hyperbaric oxygen therapy.⁷ Comparisons of different series are difficult, of course, since the prognosis for gas gangrene depends at least in part on the aetiology but these studies put the Amsterdam figures⁷ in their correct perspective.

Like many others, I believe that hyperbaric oxygen may have a place in the management of gas gangrene but there is still no acceptable clinical information which would confirm this impression. The Amsterdam workers have the facilities and the numbers of patients to provide unequivocal proof of the value of hyperbaric oxygen based on randomized studies of patients treated under the same conditions of intensive care with or without hyperbaric oxygenation.—I am, etc.,

T. T. IRVIN

Department of Surgery,
University of Leeds,
Yorkshire

- 1 Kelly, H. G., and Pace, W. G., *Surgical Forum*, 1963, 14, 46.
- 2 Glover, J. L., Mendelson, Janice, and Heitkamp, D. H., *Journal of Trauma*, 1964, 4, 462.
- 3 Hill, G. B., and Osterhout, S. S., in *Proceedings of the Third International Conference on Hyperbaric Medicine*, eds. I. W. Brown, and B. G. Cox, Washington, National Academy of Sciences, 1966.
- 4 Irvin, T. T., Moir, E. R. S., and Smith, G., *Surgery Gynecology and Obstetrics*, 1968, 127, 1058.
- 5 Gledhill, W. C., *Lancet*, 1945, 2, 264.
- 6 Altmeier, W. A., in *Proceedings of the Third International Conference on Hyperbaric Medicine*, eds. I. W. Brown, and B. G. Cox, Washington National Academy of Sciences, 1966.
- 7 Roding, B., Groeneveld, P. H. A., and Boerema, I., *Surgery Gynecology and Obstetrics*, 1972, 134, 579.

Cataracts after Renal Transplantation

SIR,—We have had three cases of steroid cataract after 39 kidney transplantations carried out in our department up to last April—an incidence of 7.6%. The patients, two men and a woman, were aged 34–42. None had received steroids or any other immunosuppressive drugs before the transplantation. All had been maintained on periodic haemodialysis for periods of 11, 12, and 1 month respectively, during which their serum calcium was over 8 mg/100 ml.

The total doses of prednisone they had received postoperatively when their cataracts appeared were 17,160, 9,700, and 7,800 mg respectively and the duration of treatment had been 780, 145, and 239 days. The average daily dose of prednisone was 22, 66, and 32 mg. In all three cases the cataract was posterior subcapsular—bilateral in two and unilateral in one.

The three patients received our standard steroid and immunosuppressive treatment. During the first postoperative month we give prednisone 1.2–5 mg/kg and azathioprine 1.5–2 mg/kg daily. After the first month, provided renal function remains stable and there is no evidence of rejection, we lower the prednisone dose to 0.5–1 mg/kg/48 hr and continue with azathioprine 1.5–2 mg/kg/24 hr. Two of our patients had an acute rejection episode during the first 40 days and they were treated with extra doses of steroids (prednisone 700 mg/24 hr intravenously for 3 and 4 days respectively) and actinomycin C (200 µg/24 hr for 2 and 3 days).

The incidence of steroid cataract in these patients seems high compared with the incidence in those with idiopathic nephrotic syndrome or chronic glomerulonephritis treated with steroids and immunosuppressive drugs, in whom we have never seen steroid cataract though their treatment sometimes lasts longer and their total dose of prednisone is higher. The early appearance of steroid cataract in the transplant patients is also remarkable. We think it possible that the large doses of prednisone given in cases of acute rejection may have a specially toxic effect to the lens.—We are, etc.,

VICTOR G. MILLET
S. CASADO PÉREZ
J. ALVAREZ GRANDE
L. HERNANDO AVENDAÑO

Nephrology Department,
Clínica de la Concepción,
Madrid, Spain

M.I.F. Test in Halothane Jaundice

SIR,—Attention has been once more drawn to the fact that jaundice after exposure to halothane, particularly in repeated doses over short periods, may be a hypersensitivity reaction.¹ Use of lymphocyte transformation tests for detecting halothane hypersensitivity has been reported and the authors suggested that other *in vitro* tests of lymphocyte function may be of value.^{1,2,3}

We have used the macrophage migration inhibition (M.I.F.) test in an investigation of two patients who developed jaundice after halothane anaesthesia. The first was a woman aged 53 who developed jaundice on the eleventh day after the second of two halothane anaesthetics within a two-week period. The second, a man aged 60,

received two halothane anaesthetics in a period of seven days and became jaundiced during the week following. Australia antigen was not detected in blood samples obtained from either patient within two weeks of the

development of jaundice. Blood was also obtained from 10 apparently healthy subjects with no history of jaundice or exposure to halothane.

Halothane suspensions were prepared as described by Paronetto and Popper³ and tested at concentrations of 1, 5, 15, and 25 µl. Lymphocytes were separated from 20 ml of heparinized blood, exposed to the "antigen" for 72 hours, and the presence of M.I.F. detected by the use of unsensitized, oil-induced, peritoneal guinea-pig macrophages in Mackness-type chambers. All tests were performed in duplicate and examined blind. The results are expressed as a mean migration index (M.I.=migration with antigen/migration without antigen; positive results show value of < 0.8).⁴

Our findings of positive results in both patients as compared to control subjects supported our suspicion that the jaundice was the result of sensitization to halothane (Fig. 1) and encourages us to continue our investigations, which will be reported in detail later.—We are, etc.,

W. JONES WILLIAMS
E. PIOLI
J. N. HORTON
A. WALDRON

Departments of Pathology and Anaesthetics,
Welsh National School of Medicine,
Cardiff

- 1 Doniach, D., *New England Journal of Medicine*, 1970, 283, 315.
- 2 Carney, F. M. T., and Van Dyke, R. A., *Anesthesia and Analgesia Current Researches*, 1972, 51, 135.
- 3 Paronetto, F., and Popper H., *New England Journal of Medicine*, 1970, 283, 277.
- 4 Jones Williams, W., Pioli, E., Jones, D. J., and Dizhero, H., *Journal of Clinical Pathology*, 1972, in press.

Defence Society Subscriptions

SIR,—Dr. Lucy Turner's letter (16 September, p. 702) calls for a reply. The Medical Defence Union has not introduced a "low-risk" category of membership, as Dr. Turner has suggested, but a "non-clinical" form of membership with a reduced sub-